IDENTIFICATION OF VASCULAR PARAMETERS BASED ON THE SAME PRESSURE PULSES WAVES USED TO MEASURE PULSE WAVE VELOCITY

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***Abstract*- Pulse Wave Velocity (PWV) is a diagnosis method to evaluate the global behavior of arterial parameters and has being used as an indicator of arterial stiffness. It is a non-invasive method based on the acquisition of pressure pulses waveforms on two sites of an arterial segment. In this paper it will be shown that based on a three-element Windkessel model and the same pressure pulse waves acquired to calculate PWV, it is possible to estimate the compliance (*C*) and associated peripheral resistance (*R*) of arterial segment under investigation. Since the Arterial Hypertension (AH) can be a result of an increased *R* value and/or decreased *C*, the estimation of *C* and *R* allows the clinicians to prescribe specific treatment to each specific condition. The estimated values of such parameters by the procedure describe in the present paper agree with the values previously reported in the literature. PWV values showed good correlation (Pearson coefficient ≥0.7) with the estimated parameters, pointing out that they can be used together to supply the clinician more information than PWV alone.**

***Keywords*- Pulse Wave Velocity, Compliance, Peripheral Resistance, System Identification.**

I. INTRODUCTION

Cardiovascular disease is one of the most important causes of death in industrialized countries [1]. It is known that the stiffing of arterial wall is normally associated with the beginning or the progression of atherosclerosis and/or Arterial Hypertension (AH) [2] and that due the insidious nature of these diseases, the early recognize of this condition permit to retard the pathologic process.

The Pulse Wave Velocity (PWV) is a non-invasive, fast and cheap diagnosis technique that can detect changes in global parameters of artery [1]. PWV is obtained from two pressure pulses waveforms, one called proximal pulse and the other distal pulse, collected in an arterial segment and is calculated as the ratio between the distance from the proximal to the distal sites to the time the pressure pulse takes to travel between them. The same characteristic points of the pressure pulse waves are taken to derived the delay time, being the "foot" of the pressure pulse contour the normally adopted in the literature [3].

Several works have shown that the PWV can be applied in the prognostic of cardiovascular disease, and also as indicator of morbidity and mortality [2, 4, 5]. For example, the brachial-radial PWV (BR-PWV) in normal subjects ranges from 6.16 to 10.95 m/s [6, 7], and consequently subjects with higher values have a prognostic of some arterial disease.

Due to the haemodynamics properties of the arterial system, higher values of PWV are associated with the developing of AH. Those values can be associated to the decrease of the arterial compliance (*C*) and/or the increase of vessel resistance (*r*) and/or the associated peripheral resistance value (*R*). It is obviously important to distinguish the primary cause for a high PWV value since the clinical treatment must differ depending on which parameter is out of the normal range.

The aim of this paper is to evaluate the compliance, the vessel resistance and the associate peripheral resistance in the brachial-radial arterial segment from a model of such segment and the same two pressure pulse waveforms acquired to measure the PWV. The estimated values of such parameters show good correlation with the respective PWV values in two groups volunteers, pointing out that they can be clinically used to give more information than PWV value alone.

II. METHODOLOGY

A system to PWV and PWA (Pulse Wave Analysis) diagnosis has been developed in the Biomedical Instrumentation Laboratory of Biomedical Engineering Program at the Federal University of Rio de Janeiro in cooperation with the Hypertension Division of the Cardiology Institute Laranjeiras, both in Rio de Janeiro, Brazil. The system has been developed in LabVIEW and the PWV and PWA results have agree very well with the literature [6, 7]. The user interface of such system can be seen in fig. 1.

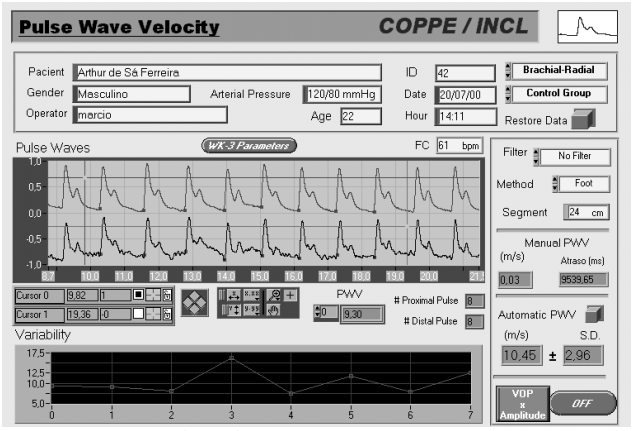

Fig. 1. The users interface of the developed PWV/PWA system. See the text for more details.

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Abstract

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In order to increase the usefulness of the mentioned system a new modulus to estimate the compliance and peripheral resistance of the studied arterial segment was designed. To perform the design a three-element Windkessel model (WK3) initially described the arterial system [8, 9].

In such model the distal pressure pulse wave was interpreted as the output, being the proximal pressure pulse wave the input. Such interpretation is illustrated in fig. 2. If the proximal pulse wave is applied to the model, the modeling parameters must determine the output pressure pulse wave. It is not difficult to demonstrate that the impulse response *h(t)* of the WK3 illustrated in fig. 2 is given by

$$h(t) = K \cdot exp((r+R)/(r \cdot R \cdot C) \cdot t) \cdot 1/(r \cdot C) \qquad (1)$$

The constant *K* was included in (1) in order to scale the proximal to distal pulses. This was done due to the fact that piezoelectric pressure transducers used to acquire the pulse waveforms don't supply an absolute pressure value and considering that proximal pressure pulses have lower amplitude than distal pressure pulses [1]. It must be mentioned that calibrated pressure pulses are not necessary to calculate the PWV value, since only the delay time between the proximal and distal pulse is used in its definition.

With the impulse response shown in (1) and an experimental version of the proximal pressure pulse wave, a theoretical output wave (theoretical distal pulse wave) can be calculated from the convolution of the proximal wave and *h(t)*. This theoretical output wave can be compared with its experimental counterpart and, through an optimization algorithm, the values of *C*, *R*, *r* and *K* can be obtained.

Following the above concept a new modulus (fig. 3) was designed and included to the pre-developed PWV/PWA system. The implemented optimization algorithm was based on a steepest descend gradient method, being the initial values to the WK3-model parameters: $C = 6.22 \times 10^{-7}$ cm^5/dyn [10]; $r = 1.15 \times 10^5$ dyn.s/cm^5; $R = 6.81 \times 10^5$ dyn.s/cm^5 and $K = 10^{-2}$. The *r* value was obtained by Poiseuille's equation [11] from the radial artery radius [10] and the *R* value from [12]. The *C* value was calculated from the radial artery cross-sectional compliance and the mean superficial length of the forearm of all subjects [10].

Using the new developed system the estimation of the parameters of the WK3 model for 2 group of subject were obtained and correlated with the values of PWV by Pearson coefficient. Values are shown in the form mean ± standard deviation.

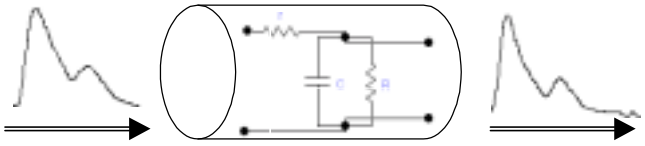

Fig. 2. Three-element Windkessel modeling the radial arterial segment, with the proximal pulse input and the distal pulse output.

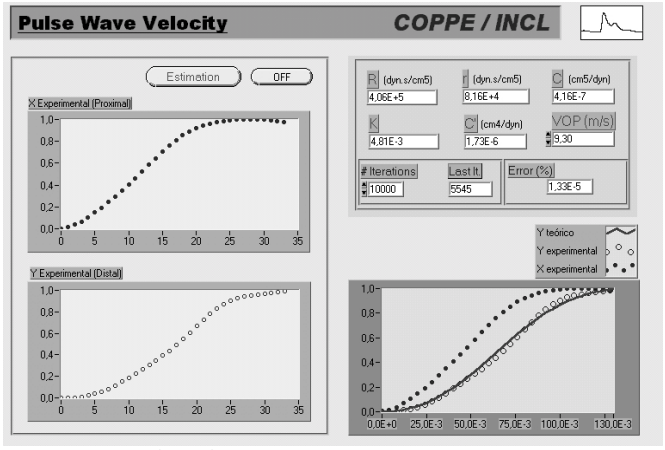

Fig. 3. User interface of the parameter extraction modulus. The right bottom sub-graphic shows the experimental proximal pulse (filled circle), the theoretical distal pulse (empty circle) and its experimental counterpart (slashed line). See the text to more details.

A. Subjects

The experimental PWV data and their respective proximal and distal pressure waveforms were obtained in two groups of volunteers: the control group (CG) and the arterial hypertension group (AHG). Because the pathologic alterations caused by the AH are a diffuse process we studied the segment of brachial to radial arteries, since it is one of the most superficial and linear arterial segment of the human body [2]. The experiments were performed in the Hypertension Division of the Cardiology Institute Laranjeiras (RJ, Brazil), being the volunteers previously informed to the aim of the experiment and have given their written agreement. The CG is represented by 12 subjects (10 men and 2 women, 28.08 age ± 3.96 years), and the AHG is compounded by 12 subjects (8 men and 4 women, age 38.50 ± 10.04 years).

The inclusion criteria in the CG were: normal ECG, arterial pressure lower than 130/88 mm Hg, age under 40 years, no familiar history of premature heart attack, BR-PWV under 10.95 m/s.

During the acquisition of the pressure pulse waveforms the subjects were in supine position, with the arms beside the thorax, being a rest time of 5.0 min always observed before the acquisition. Proximal site to the acquisition was in the anterior region of the elbow over the brachial artery and the distal site in the anterior region of the wrist over the radial artery.

B. Data Acquisition

The acquisition of the pressure pulses waveforms was performed with piezoelectric transducers (HP 21050-A, bandwidth 0,01 Hz to 2 kHz) connected to preamplifiers specially developed for this study, that imposes a lowpass frequency limitation of 3.38 Hz. The two pressure pulse waveforms signals were digitalized by a 12 bits, 16 single-ended channels, Fs = 100 Ks/s acquisition board, model AT-

MIO 16 - National Instruments. Although the signals basic band was limited to 3.38Hz, the signals were acquired at a sample frequency of 2 kHz in order to increase the time resolution and consequently the accuracy of the PWV value.

III. Results

The numeric values of PWV and the estimated values of the WK3 model for the CG can be seen in table I. The AHG results were separated in two subgroups, those with main alterations in R value (AHG-R, shown in table II) or C value (AHG-C, table III).

The Pearson coefficient between the vascular parameters and PWV are shown in table IV.

IV. Discussion

The results of PWV differ statistically between the CG and AHG (7.45 ± 1.62 m/s and 21.78 ± 7.19 m/s, respectively, $p<0.01$) but not between AHG-R and AHG-C (19.57 ± 5.89 m/s and 23.99 ± 8.20 m/s, respectively, $p<0.18$), being PWV higher for the AHG. The stiffener the arterial walls, faster is the velocity of propagation of the pulses.

The estimated compliance values (ranging from 4.43 to 7.75 x 10^{-7} cm^5/dyn) are not statically different between the CG (5.74 ± 1.44 x 10^{-7} cm^5/dyn) and the AHG as a whole (4.90 ± 2.02 x10^{-7} cm^5/dyn). However, there are statistically difference between the CG and AHG-C in such parameter (5.74 ± 1.44 x 10^{-7} cm^5/dyn and 3.01 ± 0.68 x 10^{-7} cm^5/dyn, respectively, $p<0.01$). Being the stiffening a general process, a muscular arterial segment with this pathologic process reflex the general condition of the arterial system.

The values of the associated peripheral resistance are statically greater ($p<0.01$) in the AHG-R (6.16 ± 1.83 x 10^5 dyn.s/cm^5), but not in the AHG-C (3.81 ± 0.68 x 10^5 dyn.s/cm^5), when compared with the CG (4.50 ± 1.04 x 10^5 dyn.s/cm^5). By the other hand, the values of compliance of the AHG-C (3.01 ± 0.68 x 10^{-7} cm^5/dyn) are statistically lower ($p<0.01$) than the AHG-R (6.78 ± 0.13 x 10^{-7} cm^5/dyn). This data comprise that arterial hypertension is due a balanced interaction between *C* and *R*, where one or both parameters can be out of normality.

The values of the vessel resistance (*r*) do not showed statistically difference between the two groups (CG = 1.11 ± 0.25 x 10^5 dyn.s/cm^5; AHG = 1.39 ± 1.46 x 10^5 dyn.s/cm^5, $p<0.25$), indicating that there are not a atherosclerosis processes initialized.

Table III shows that PWV values in the control (normal) group showed good correlation with the compliance (r = -0.72), the associated peripheral resistance (r = -0.73) and the resistance of the arterial studied segment (r = -0.75), pointing out all of them are really important to the PWV values.

TABLE I
EXTRACTED PARAMETERS FOR THE NORMAL SUBJECTS GROUP

G	BR PWV	*R*	*C*	*R*
M	6.45	3.97	6.33	1.23
M	6.93	4.22	7.05	1.32
M	7.90	3.96	4.43	0.87
M	7.69	4.09	6.62	1.26
M	8.59	4.04	5.25	1.07
M	7.93	5.59	7.51	1.40
M	7.49	4.13	4.92	1.01
M	9.58	3.87	3.98	0.80
M	10.47	3.03	3.16	0.62
M	5.70	4.68	6.30	1.19
F	5.70	5.54	5.56	1.13
F	4.96	6.91	7.75	1.42
ME	7.45	4.50	5.74	1.11
SD	1.62	1.04	1.44	0.25

Where G: gender; M: male; F: female; BR PWV: brachial-radial pulse wave velocity (m/s); r: vessel resistance (10^5 dyn.s/cm^5); R: peripheral resistance (10^5 dyn.s/cm^5); C: vessel compliance (10^{-7} cm^5/dyn); ME: mean; SD: standard deviation.

TABLE II
EXTRACTED PARAMETERS FOR THE ARTERIAL HYPERTENSION SUBJECTS SUBGROUP (AHG-R)

G	BR PWV	*R*	*C*	*r*
M	29.91	9.79	6.98	1.29
M	22.58	5.38	6.68	1.26
M	14.50	4.97	6.80	1.27
F	17.29	5.74	6.61	1.24
F	18.66	6.10	6.82	1.27
F	14.50	4.97	6.80	1.27
ME	19.57	6.16	6.78	1.27
SD	5.89	1.83	0.13	0.02

Where G: gender; M: male; F: female; BR PWV: brachial-radial pulse wave velocity (m/s); r: vessel resistance (10^5 dyn.s/cm^5); R: peripheral resistance (10^5 dyn.s/cm^5); C: vessel compliance (10^{-7} cm^5/dyn); ME: mean; SD: standard deviation.

TABLE III
EXTRACTED PARAMETERS FOR THE ARTERIAL HYPERTENSION SUBJECTS SUBGROUP (AHG-C)

G	BR PWV	*R*	*C*	*r*
M	24.58	4.29	3.40	0.71
M	16.71	4.47	2.91	0.67
M	37.61	3.87	4.13	0.88
M	17.83	4.19	2.64	0.53
M	29.05	3.38	2.84	5.90
F	18.17	2.67	2.15	0.42
ME	23.99	3.81	3.01	1.52
SD	8.20	0.68	0.68	2.15

Where G: gender; M: male; F: female; BR PWV: brachial-radial pulse wave velocity (m/s); r: vessel resistance (10^5 dyn.s/cm^5); R: peripheral resistance (10^5 dyn.s/cm^5); C: vessel compliance (10^{-7} cm^5/dyn); ME: mean; SD: standard deviation.

TABLE IV
PEARSON COEFFICIENT BETWEEN PRESSURE PULSE, PWV AND THE ARTERIAL PARAMETERS EXTRACTED WITH WK3

Group	BR PWV/*R*	BR PWV/*C*	BR PWV/*r*
CG	-0.73	-0.72	-0,75
AHG-R	0.89	0.51	0.51
AHG-C	-0.06	0.81	0.36

Where BR PWV: brachial-radial pulse wave velocity (m/s); r: vessel resistance (10^5 dyn.s/cm^5); R: peripheral resistance (10^5 dyn.s/cm^5); C: vessel compliance (10^{-7} cm^5/dyn); ME: mean; SD: standard deviation;

V. Conclusion

The new modulus of the PWV/PWA system was able to estimate values of arterial parameters (*R*, *C* and *r*) that are in agreement with the values reported in literature for normotensive subjects and patients with AH [10, 12]. These parameters reflect the capability of the radial artery to accommodate the blood volume transmitted by the arterial circuit through the thorax and arm in order to maintain constant the blood flow to the periphery (the hand). Alteration in one or more of these parameters can cause an unbalance on this capability, resulting on increased mean arterial pressure.

PWV has been reported since 1922 [5] as a good indicator of pathologic process in the arterial system. Despite this fact, PWV values can not alone supply separated information concerning the main changes in the arterial properties. Since the compliance and the vessel resistance, as well as the peripheral associated resistance values are the properties that govern the arterial pulse waveforms, different combinations of these values can result in similar, higher of normal range, PWV values. The presented method to estimate the arterial properties, in a non-invasive way and based on the same pulse waveforms acquired to calculate PWV, seems to be able to supply coherent parameters that can help the clinician to better orient his/her procedure in the patient treatment. Obviously the number of the cases must be increase to support the above guess, being such study actually in curse.

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